

=> d his

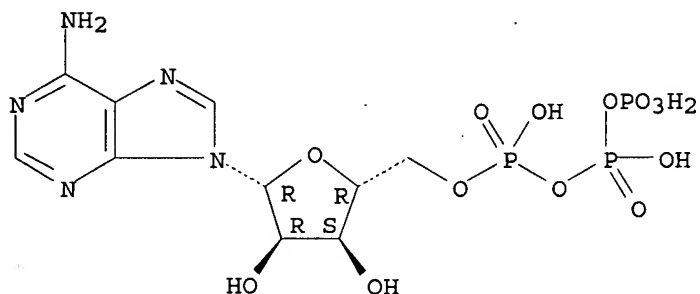
(FILE 'HOME' ENTERED AT 17:53:01 ON 21 JUN 2007)

FILE 'CAPLUS, MEDLINE' ENTERED AT 17:53:14 ON 21 JUN 2007

L1	1 S PERITONEAL INJUR? (P) ATP
L2	0 S PERITONEAL INJUR? (P) ADENOSINE TRIPHOSPHATE?
L3	803 S CELL INJUR? (P) ATP
L4	0 S CELL INJURY? (P) ATP (P) DIALYSIS
L5	0 S PERITONEAL CELL INJURY? (P) ATP (P) DIALYSIS
L6	0 S PERITONEAL CELL INJURY? (P) ATP
L7	0 S PERITONEAL CELL INJUR? (P) ATP
L8	0 S PERITON? CELL INJUR? (P) ATP
L9	0 S PERITON? CELL INJUR? (P) ADENOSINE TRIPHOSPHATE
L10	0 S PERITON? CELL INJUR? (P) NUCLEOTIDE?
L11	169 S CELL INJUR? (P) NUCLEOTIDE?
L12	42 S CELL INJUR? (P) NUCLEOTIDE? (P) TREAT?
L13	0 S L12 AND DIALYSIS
L14	0 S L12 AND DIAL?
L15	63 S CELL INJUR? (P) ADENINE NUCLEOTIDE?
L16	52 S L15 NOT L12
L17	1 S L16 AND PATIENT?
L18	0 S L16 AND ADMINISTER?
L19	106 S L11 NOT L15
L20	106 S L19 NOT L16
L21	75 S L19 NOT L12
L22	19 S CELL INJUR? (P) ATP (P) PATIENT?

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN  
 RN 56-65-5 REGISTRY  
 ED Entered STN: 16 Nov 1984  
 CN Adenosine 5'-(tetrahydrogen triphosphate) (CA INDEX NAME)  
 OTHER NAMES:  
 CN 5'-ATP  
 CN Adenosine 5'-triphosphate  
 CN Adenosine 5'-triphosphoric acid  
 CN Adenosine triphosphate  
 CN Adenosine, 5'-(tetrahydrogen triphosphate)  
 CN Adenylpyrophosphoric acid  
 CN Adephos  
 CN Adetol  
 CN Adynol  
 CN Atipi  
 CN ATP  
 CN ATP (nucleotide)  
 CN Atriphos  
 CN Cardenosine  
 CN Fosfobion  
 CN Glucobasin  
 CN Myotriphos  
 CN Phosphobion  
 CN Striadyne  
 CN Triadenyl  
 CN Triphosphaden  
 CN Triphosphoric acid adenosine ester  
 FS STEREOSEARCH  
 DR 896506-78-8, 10168-83-9, 16488-07-6, 51569-41-6, 71800-44-7, 84412-18-0  
 MF C10 H16 N5 O13 P3  
 CI COM  
 LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN\*,  
 BIOSIS, BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS,  
 CHEMLIST, CIN, CSCHM, CSNB, DDFU, DETHERM\*, DRUGU, EMBASE, GMELIN\*,  
 IFICDB, IFIPAT, IFIUDB, IMSDRUGNEWS, IMSRESEARCH, IPA, MEDLINE, MRCK\*,  
 NAPRALERT, PHAR, PIRA, PROMT, PS, RTECS\*, SCISEARCH, SPECINFO,  
 TOXCENTER, TULSA, USPAT2, USPATFULL  
 (\*File contains numerically searchable property data)  
 Other Sources: DSL\*\*, EINECS\*\*, TSCA\*\*  
 (\*\*Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



\*\*PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT\*\*

78925 REFERENCES IN FILE CA (1907 TO DATE)  
 1609 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA  
 79056 REFERENCES IN FILE CAPLUS (1907 TO DATE)  
 19 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L6 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:453073 CAPLUS

DOCUMENT NUMBER: 140:429098

TITLE: Peritoneal dialysis method with solution containing ATP

INVENTOR(S): Kiribayashi, Kei; Yorioka, Noriaki

PATENT ASSIGNEE(S): Kowa Co., Ltd., Japan

SOURCE: PCT Int. Appl., 21 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004045679	A1	20040603	WO 2003-JP14790	20031120
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
CA 2506704	A1	20040603	CA 2003-2506704	20031120
AU 2003284594	A1	20040615	AU 2003-284594	20031120
EP 1563858	A1	20050817	EP 2003-774083	20031120
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK			
CN 1713926	A	20051228	CN 2003-80103652	20031120
NZ 539796	A	20061130	NZ 2003-539796	20031120
US 2006019925	A1	20060126	US 2005-533538	20050502
PRIORITY APPLN. INFO.:			US 2002-427980P	P 20021121
			WO 2003-JP14790	W 20031120

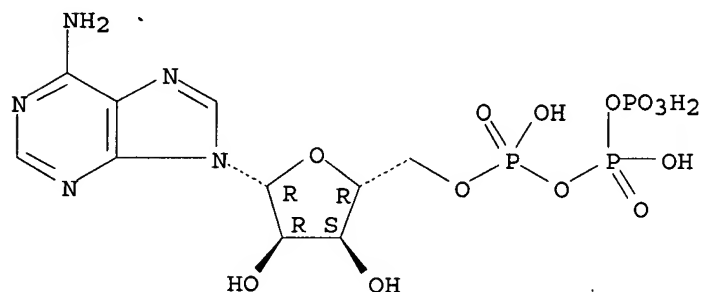
AB Disclosed are a peritoneal dialyzate fluid containing ATP or its salt and a peritoneal dialysis method using the same. This peritoneal dialyzate fluid is highly safe and causes no peritoneal injury even employed over a long time. The effect of ATP on protection of human peritoneal mesothelial cells (HPMC) from high concentration of glucose in the culture medium was examined

IT 56-65-5, ATP, biological studies

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses) (peritoneal dialysis method with solution containing ATP)

RN 56-65-5 CAPLUS

CN Adenosine 5'-(tetrahydrogen triphosphate) (CA INDEX NAME)



Absolute stereochemistry.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1962:69554 CAPLUS

DOCUMENT NUMBER: 56:69554

ORIGINAL REFERENCE NO.: 56:13442d-h

TITLE: Postoperative electrolyte balance

AUTHOR(S): Carstensen, E.; Scheibe, O.

CORPORATE SOURCE: Chirurg. Univ.-Kiln., Hamburg/Eppendorf, Germany

SOURCE: Deutsche Medizinische Wochenschrift (1962), 87,

394-400

CODEN: DMWOAX; ISSN: 0012-0472

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

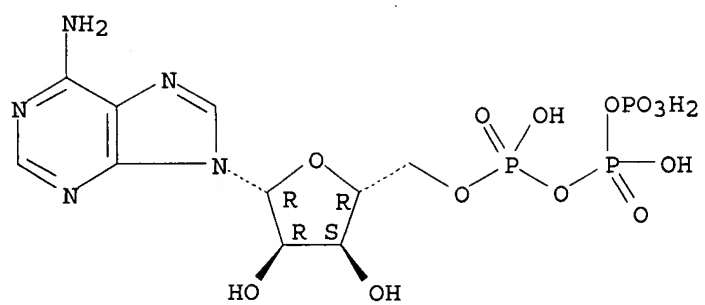
AB In 132 patients undergoing lung, thyroid, stomach, or colonic resections, cholecystectomy, or mammary amputations electrolyte and H<sub>2</sub>O balance studies were carried out from the start of each operation to the end of the 6th postoperative day; results were statistically analyzed. Na balance was pos. and Na was retained up to the 4th day, regardless of the type of operation; K balance was neg. and K was excreted in abnormally large amts. up to the 3rd day. Postoperative administration of physiol. NaCl favored Na retention and increased K excretion. The K excretion was independent of the total N elimination, indicating that the K did not derive from tissue cellular disintegration. The K level in erythrocytes was significantly lower in the patients than in normal blood donors up to the 2nd day, at which time the level was at its lowest. It was concluded that at least part of the increased output of K derived from erythrocytes which, together with the K in the other tissues comprising the intracellular space, normally acted as a buffer for the extracellular space whose K concentration gradient was essential for the maintenance of the cell membrane potential. During surgery, venous blood HCO<sub>3</sub><sup>-</sup> tended to decrease. Urinary pH remained between 5.0 and 6.0 throughout the entire exptl. period; titratable acidity was greatest during the 1st 48 hrs. and the urine volume/hr. fluctuated in parallel with titratable acidity, being lowest on the day of surgery (equivalent to about 0.5 the fluid intake). Plasma cortisol increased significantly until the 3rd day, being highest 4 hrs. postoperatively (3-4 times the average normal value of 11.2 γ%); this abnormal increase might have been the cause of the electrolyte disturbances observed. In 29 patients who received no postoperative sugar infusions, the adenosine triphosphate (ATP) level decreased and was lowest by the 2nd day; where infusions of glucose or fructose were administered the ATP curve was fundamentally different. It was concluded that postoperative complications, such as ileus, peritonitis, or vomiting, might lead to the exhaustion of the intracellular buffering mechanism and necessitate substitution treatment with K-containing solns.

IT 56-65-5, Adenosine triphosphate  
(in blood plasma after surgery)

RN 56-65-5 CAPLUS

CN Adenosine 5'-(tetrahydrogen triphosphate) (CA INDEX NAME)

Absolute stereochemistry.



L11 ANSWER 12 OF 24 MEDLINE on STN  
 ACCESSION NUMBER: 2005639081 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 16215382  
 TITLE: Gut luminal microdialysis of glycerol as a marker of intestinal ischemic injury and recovery.  
 AUTHOR: Solligard Erik; Juel Ingebjorg S; Bakkelund Karin; Jynge Per; Tvedt Kare E; Johnsen Harald; Aadahl Petter; Gronbech Jon Erik  
 CORPORATE SOURCE: Department of Anesthesiology and Intensive Care, St. Olav University Hospital, Norwegian University of Science and Technology, N-7006 Trondheim, Norway..  
 erik.sollegard@ntnu.no  
 SOURCE: Critical care medicine, (2005 Oct) Vol. 33, No. 10, pp. 2278-85.  
 Journal code: 0355501. ISSN: 0090-3493.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LANGUAGE: English  
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
 ENTRY MONTH: 200512  
 ENTRY DATE: Entered STN: 3 Dec 2005  
 Last Updated on STN: 24 Dec 2005  
 Entered Medline: 23 Dec 2005

AB OBJECTIVE: To evaluate microdialysis as a method to assess different degrees of intestinal damage and recovery during ischemia and reperfusion; to evaluate information obtained from microdialysis catheters in the peritoneum, the gut wall, and the gut lumen. DESIGN: Randomized, controlled animal experiment. SETTING: University laboratory animal center. SUBJECTS: Twenty-seven domestic pigs. INTERVENTIONS: The superior mesenteric artery was cross-clamped for 60 mins (n = 14) or 120 mins (n = 10) followed by 2 or 4 hrs of reperfusion. Three pigs served as controls. MEASUREMENTS AND MAIN RESULTS: Intestinal mucosal integrity was assessed by morphometry, adenosine triphosphate in the gut wall, and permeability of C-polyethylene glycol. Lactate, glycerol, pyruvate, and glucose were measured by microdialysis. Changes in adenosine triphosphate, permeability, or lactate did not correlate to different extents of intestinal damage caused by 60 or 120 mins of ischemia. During the reperfusion period, pigs with 60 mins of intestinal ischemia showed a faster recovery of these variables than pigs with 120 mins of intestinal ischemia. Glycerol increased with increasing duration of the ischemic insult. After 60 mins of intestinal ischemia, glycerol in the gut lumen decreased toward baseline but remained high after 120 mins of intestinal ischemia. There was a good correlation between gut luminal glycerol and recovery of mucosal damage throughout the reperfusion period. In the peritoneal cavity, both glycerol and lactate decreased to baseline relatively shortly after onset of reperfusion independent of the duration of intestinal ischemia. CONCLUSIONS: Microdialysis of glycerol provides information about the extent and severity of intestinal damage after ischemia and about the ensuing recovery. The gut lumen is to be preferred as a site for placement of microdialysis catheters.

L11 ANSWER 13 OF 24 MEDLINE on STN  
 ACCESSION NUMBER: 2004069220 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 14870882  
 TITLE: The in vitro biocompatibility performance of a 25 mmol/L bicarbonate/10 mmol/L lactate-buffered peritoneal dialysis fluid.  
 AUTHOR: Skoufos Line; Topley Nicholas; Cooker Laurinda; Dawnay Anne; Millar David J; Holmes Clifford J; Faict Dirk  
 CORPORATE SOURCE: Baxter Healthcare Corporation, McGaw Park, Illinois 60085, USA.. line\_skoufos@baxter.com  
 SOURCE: Kidney international. Supplement, (2003 Dec) No. 88, pp.

S94-9.

Journal code: 7508622. ISSN: 0098-6577.

PUB. COUNTRY: United States  
DOCUMENT TYPE: (COMPARATIVE STUDY)  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200407  
ENTRY DATE: Entered STN: 12 Feb 2004  
Last Updated on STN: 29 Jul 2004  
Entered Medline: 28 Jul 2004

AB The in vitro biocompatibility performance of a 25 mmol/L bicarbonate/10 mmol/L lactate-buffered peritoneal dialysis fluid. BACKGROUND: The biocompatibility profile of a new peritoneal dialysis (PD) solution (Physioneal 35) was determined using a selection of in vitro assay systems. Physioneal 35 is buffered by a combination of 25 mmol/L bicarbonate and 10 mmol/L lactate, thereby providing a solution with a total of 35 mmol/L of alkali to complement the currently available 25 mmol/L bicarbonate and 15 mmol/L lactate combination solution, Physioneal 40. In addition, the new solution contains a calcium concentration of 1.75 mmol/L rather than 1.25 mmol/L present in Physioneal 40. Physioneal 35 and 40 are manufactured in double chamber bag systems that permit separation of glucose from the buffer during sterilization. When the two chambers are mixed just before patient use, the resulting solution has a neutral pH and reduced glucose degradation content. Physioneal 35 was evaluated for its cytotoxicity potential using a murine fibroblast assay, its acute effect on human neutrophil and human peritoneal mesothelial cell function, and its in vitro potential to form advanced glycation end products (AGE). The biocompatibility characteristics of this new formulation were compared with that of a conventional, lactate-based solution and to that of its parent formulation, Physioneal 40. METHODS: Proliferation of murine fibroblasts was determined after exposure to dialysis fluids for 72 hours. Cell viability was assayed by the ability to take up neutral red dye. Human neutrophils were exposed for 15 minutes to dialysis fluids, and their ATP content and phorbol 12-myristate 13-acetate (PMA) stimulated chemiluminescence response was determined as a measure of viability and respiratory burst activity, respectively. Cellular interleukin (IL)-1 $\beta$ -driven IL-8 synthesis by human mesothelial cells following acute exposure to dialysis fluids was also assessed. Advanced glycation end product formation in the dialysis fluids was measured after 5 and 20 days of incubation with human serum albumin (HSA) as the model protein. RESULTS: In all assays employed, the biocompatibility profile of Physioneal 35 was similar to that of the Physioneal 40 parent formulation. Physioneal 35 showed a significant improvement in biocompatibility performance compared to a pH neutralized conventional lactate-buffered peritoneal dialysis solution in the murine fibroblast assay. In the acute exposure assays, human neutrophil viability and respiratory burst were significantly improved compared with the acidic, conventional solution; however, no statistically significant improvement were seen in mesothelial cells. AGE formation, which is thought to be an important mechanism by which glucose and glucose degradation products cause structural and functional changes of the peritoneal membrane, was significantly lower in Physioneal 35 compared with the conventional dialysis solution. CONCLUSION: The biocompatibility profile of Physioneal 35 was similar to that of the original Physioneal 40 bicarbonate/ lactate-buffered dialysis solution, confirming that differences in both buffer content and calcium concentration do not affect biocompatibility performance. Both bicarbonate/lactate formulations (Physioneal 35 and Physioneal 40) were more biocompatible than a conventional lactate-buffered dialysis solution in this in vitro biocompatibility assessment.

ACCESSION NUMBER: 2001529960 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 11576893  
 TITLE: Peritoneal dialysis fluids with a physiologic pH based on either lactate or bicarbonate buffer-effects on human mesothelial cells.  
 AUTHOR: Plum J; Razeghi P; Lordnejad R M; Perniok A; Fleisch M; Fussboller A; Schneider M; Grabensee B  
 CORPORATE SOURCE: Department of Nephrology and Rheumatology, Heinrich Heine-University, Dusseldorf, Germany.. plum@uni-duesseldorf.de  
 SOURCE: American journal of kidney diseases : the official journal of the National Kidney Foundation, (2001 Oct) Vol. 38, No. 4, pp. 867-75.  
 Journal code: 8110075. E-ISSN: 1523-6838.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200110  
 ENTRY DATE: Entered STN: 1 Oct 2001  
 Last Updated on STN: 22 Oct 2001  
 Entered Medline: 18 Oct 2001  
 AB Conventional lactate (Lac)-buffered peritoneal dialysis (PD) solutions have turned out to be detrimental to human peritoneal cells, especially because of a low pH. In the present study, we focus on potential differences between Lac and bicarbonate (Bic) as a buffer when adjusted to a physiological pH. All test fluids were buffered with either 40 mmol/L of Lac or 34 mmol/L of Bic, sterile filtered, and adjusted to a pH of 7.4. Osmotic agents used were 1.36% glucose (Glu), 3.86% Glu, 1% amino acids (AA), and 7.5% Glu polymer (Glupoly). Human peritoneal mesothelial cells (HPMCs) were isolated from the omentum majus, grown to confluence, and incubated after the second passage for 15 minutes (37 degrees C and 5% carbon dioxide) with the test fluids. Cytotoxicity was controlled by measuring apoptotic and necrotic cells with cytofluorometry. Aerobic cell metabolism (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide [MTT] assay) and intracellular adenosine triphosphate (ATP) concentrations were measured to assess cell viability. Release of interleukin-6 (IL-6) from HPMCs was determined as a parameter of cellular host defense. No significant difference in apoptosis or necrosis rates was found between the solutions adjusted to normal pH. However, in the MTT assay, Bic solutions were superior to corresponding Lac pendants at an identical pH of 7.4 ( $P < 0.01$ ). Intracellular ATP concentrations reflected a very similar pattern ( $P < 0.05$ ). Glupoly in combination with Lac showed an impaired pattern with both the MTT and ATP assays. Regarding IL-1beta-stimulated IL-6 release, there was a small, but not significantly better, response for Bic. Differences in manifest cell cytotoxicity reflected by apoptosis and necrosis rates could not be detected comparing PD solutions buffered with Lac or Bic at a physiological pH. However, distinct parameters of cell metabolism were superior with Bic compared with Lac. Especially Glupoly was inferior in combination with Lac as a buffer.

L11 ANSWER 15 OF 24 MEDLINE on STN

ACCESSION NUMBER: 1998449602 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 9778153  
 TITLE: Direct administration and utilization of [1-13C] glucose by fetal brain and liver tissues under normal and ischemic conditions: 1H, 31P, and 13C NMR studies.  
 AUTHOR: Brand A; Gil S; Leibfritz D; Yavin E  
 CORPORATE SOURCE: Institut fur Organische Chemie, Universitat Bremen, Germany.  
 SOURCE: Journal of neuroscience research, (1998 Oct 1) Vol. 54, No. 1, pp. 97-108.



Journal code: 7600111. ISSN: 0360-4012.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199812  
ENTRY DATE: Entered STN: 15 Jan 1999  
Last Updated on STN: 15 Jan 1999  
Entered Medline: 11 Dec 1998

AB Three distinct, maternal-independent routes (e.g. intraamniotic, intraperitoneal and intracerebral), for [1-13C]glucose utilization by fetal brain and liver tissues, were examined by multinuclear magnetic resonance (NMR) spectroscopy before and after vascular occlusion of the maternal-fetal blood flow. Labeled lactate was the major glycolytic product by all routes, but in addition labeled TCA cycle products were also generated. Fractional 13C enrichment in both glucose and lactate were always higher in the ischemic state compared to controls using either one of the three routes studied. After intraperitoneal injection total glucose in the fetal brain was decreased by 85% after 20 min reperfusion following 20 min ischemia, but was elevated up to 170% after 60 min. [1-13C]glucose increased continuously by up to 370% after 60 min. Total glucose in the fetal liver remained unchanged while [1-13C]glucose increased up to 380%. Total lactate level in brain was 50-80% above the control apart from a transient increase (140%) notable after 40 min reperfusion. The kinetics of [3-13C]lactate followed a similar time course. At the same time when lactate was transiently increased in fetal brain, total lactate as well as 13C-labeled lactate showed a transient decrease in liver after 40 min. While the ways of mobilization of energy substrates for maintaining adequate metabolic activity in the fetal brain remain still unclear, the present 13C NMR studies suggest that both liver glucose and lactate can contribute to brain metabolism particularly under ischemic stress.

L11 ANSWER 16 OF 24 MEDLINE on STN  
ACCESSION NUMBER: 93171622 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 8436827  
TITLE: Lactate transport in macrophages.  
AUTHOR: Loike J D; Kaback E; Silverstein S C; Steinberg T H  
CORPORATE SOURCE: Rover Physiology Laboratories, Department of Physiology and Cellular Biophysics, Columbia University College of Physicians and Surgeons, New York, NY 10032.  
CONTRACT NUMBER: AI00893 (NIAID)  
AI20516 (NIAID)  
DK39100 (NIDDK)  
SOURCE: Journal of immunology (Baltimore; Md. : 1950), (1993 Mar 1)  
Vol. 150, No. 5, pp. 1951-8.  
Journal code: 2985117R. ISSN: 0022-1767.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 199303  
ENTRY DATE: Entered STN: 2 Apr 1993  
Last Updated on STN: 3 Feb 1997  
Entered Medline: 24 Mar 1993

AB Macrophages perform phagocytic and effector activities in a number of different tissues. The environment of the inflammatory foci in which they function is often acidic and contains an abundance of lactate. We characterized the ability of thioglycollate-elicited mouse peritoneal macrophages to accumulate lactate from the medium and to use this lactate to maintain intracellular energy stores. Lactate

uptake was stereospecific for L-lactate and was inhibited by the organic anion transport blocker probenecid but not by concentrations of 4,4'-diisothiocyanatostilbene-2,2'-disulfonic acid that block anion exchangers. L-[14C]Lactate uptake was not affected by variation of the extracellular Na<sup>+</sup> concentration but was enhanced by acidification of the extracellular medium, suggesting that lactate uptake was mediated by a proton cotransport system. The enhanced accumulation of [14C]-lactate seen in medium at pH 6.0 to 6.5 was inhibited by probenecid or by an excess of unlabeled L-lactate. When macrophages were incubated in PBS without glucose for 6 h, intracellular stores of phosphocreatine were 13 nmol/mg of protein, compared with 44 nmol/mg of protein in cells incubated in medium containing glucose. When lactate was substituted for glucose, phosphocreatine stores were 32 nmol/mg of protein. These studies reveal that macrophages take up L-lactate in a pH-dependent manner and that lactate uptake occurs via a probenecid-inhibitable monocarboxylate transporter; they suggest that macrophages can utilize this lactate as an energy source.

L11 ANSWER 17 OF 24 MEDLINE on STN  
 ACCESSION NUMBER: 92110328 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 1764469  
 TITLE: Glucose and glutamine metabolism in rat macrophages: enhanced glycolysis and unaltered glutaminolysis in spontaneously diabetic BB rats.  
 AUTHOR: Wu G Y; Field C J; Marliss E B  
 CORPORATE SOURCE: McGill Nutrition and Food Science Centre, Royal Victoria Hospital, Montreal, Quebec, Canada.  
 SOURCE: Biochimica et biophysica acta, (1991 Dec 6) Vol. 1115, No. 2, pp. 166-73.  
 Journal code: 0217513: ISSN: 0006-3002.  
 PUB. COUNTRY: Netherlands  
 DOCUMENT TYPE: (IN VITRO)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199202  
 ENTRY DATE: Entered STN: 8 Mar 1992  
 Last Updated on STN: 6 Feb 1998  
 Entered Medline: 20 Feb 1992

AB Metabolism of glutamine (Gln, 2 mM) and glucose (5 mM) was studied in vitro in isolated resident peritoneal macrophages from both normal (BBn) and spontaneously diabetic BB (BBd) rats. The major products from Gln were ammonia, glutamate, CO<sub>2</sub> and to a lesser extent aspartate. Glucose decreased (P less than 0.01) the production of ammonia, CO<sub>2</sub> and aspartate from Gln by 34-60%, but had no effect on the amount of glutamate accumulated. The major products from glucose were lactate and to a much lesser extent pyruvate and CO<sub>2</sub>. Gln decreased (P less than 0.01) <sup>14</sup>CO<sub>2</sub> production from [U-<sup>14</sup>C] glucose by 19-28%, increased (P less than 0.01) pyruvate production by 35-49%, but had no effect on lactate production. The fraction of glucose metabolized via the pentose phosphate pathway (PC) was less than 5%. There were no significant differences in Gln metabolism between BBn and BBd macrophages. The production of lactate and pyruvate and the flux from glucose into the PC were increased (P less than 0.01) by 2.4, 1.8 and 1.5-fold, respectively, in BBd cells. Increased macrophage glucose metabolism was also observed in diabetes-prone BB (BBdp) rats at 75-80 days but not at 50 days of age. In the presence of both Gln and glucose, potential ATP production from glucose was 2- and 4-times that from Gln, respectively, in BBn and BBd cells. Lactate production was the major pathway for glucose-derived ATP generation. These results demonstrate (a) glycolysis and flux from glucose through the

pentose phosphate pathway are enhanced with no alteration in glutaminolysis in BBd macrophages; and (b) glucose may be a more important fuel than Gln for macrophages, particularly in BBd rats. The increased glucose metabolism may be associated with functional activation of the macrophages that have been proposed to be involved in beta-cell destruction and the development of diabetes.

L11 ANSWER 18 OF 24 MEDLINE on STN  
ACCESSION NUMBER: 92051818 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 1719516  
TITLE: Dual effect of magnesium on compound 48/80-induced histamine secretion from rat peritoneal mast cells.  
AUTHOR: Bertelsen H; Johansen T  
CORPORATE SOURCE: Department of Pharmacology, Odense University, Denmark.  
SOURCE: Pharmacology & toxicology, (1991 Jul) Vol. 69, No. 1, pp. 28-33.  
Journal code: 8702180. ISSN: 0901-9928.  
PUB. COUNTRY: Denmark  
DOCUMENT TYPE: (IN VITRO)  
Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199112  
ENTRY DATE: Entered STN: 24 Jan 1992  
Last Updated on STN: 6 Feb 1998  
Entered Medline: 23 Dec 1991

AB The effect of magnesium on the secretory response to compound 48/80 from rat peritoneal mast cells was studied. The decrease in secretion caused by calcium deprivation was enlarged by magnesium. Glucose partially counteracted the decrease caused by calcium deprivation but not the one caused by magnesium. The addition of calcium to the cells simultaneously with compound 48/80 completely restored the secretory response if magnesium was present. The response was only partially restored in a magnesium- and glucose-free medium, whereas it was almost completely restored if glucose was present. Magnesium had a considerable effect on the restoration of the secretory response of EGTA-treated cells, whereas the effect of glucose was minimal indicating that an effect on the energy metabolism was of minor importance. The secretory response could also be restored by an exposure of the cells to calcium prior to stimulation with compound 48/80. This was, however, only observed if magnesium was present and glucose had no effect. The influence of magnesium on the restoration of the secretory response may partly occur by an effect on the energy metabolism, partly by an effect on the stimulus-secretion coupling. We propose that insufficient supply of Mg<sup>2+</sup> to the G-protein during activation by compound 48/80 might cause a suboptimal signal transduction.

L11 ANSWER 19 OF 24 MEDLINE on STN  
ACCESSION NUMBER: 89374090 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 2775207  
TITLE: Rates of utilization of glucose, glutamine and oleate and formation of end-products by mouse peritoneal macrophages in culture.  
AUTHOR: Newsholme P; Newsholme E A  
CORPORATE SOURCE: Department of Biochemistry, University of Oxford, U.K.  
SOURCE: The Biochemical journal, (1989 Jul 1) Vol. 261, No. 1, pp. 211-8.  
Journal code: 2984726R. ISSN: 0264-6021.  
PUB. COUNTRY: ENGLAND: United Kingdom  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
LANGUAGE: English

FILE SEGMENT: Priority Journals  
ENTRY MONTH: 198909  
ENTRY DATE: Entered STN: 9 Mar 1990  
Last Updated on STN: 6 Feb 1998  
Entered Medline: 27 Sep 1989

AB 1. The metabolism of mouse thioglycollate-elicited peritoneal macrophages was studied in culture for up to 96 h. 2. The rates of glycolysis, lactate formation and glutamine utilization were approximately linear with time for at least 80 h of culture. 3. The rates of glucose and glutamine utilization by cultured macrophages were approx. 500 and 90 nmol/h per mg of protein respectively. This rate of glucose utilization is at least 50% greater than that previously reported for macrophages during 60 min incubation in a shaking flask; and it is now increased by addition of glutamine to the culture medium. The rate of glutamine utilization in culture is similar to that previously reported for macrophages during 60 min incubation. The major end-product of glucose metabolism is lactate, and those of glutamine metabolism are CO<sub>2</sub>, glutamate, ammonia and alanine. 4. Oleate was utilized by these cells: <sup>14</sup>C from [<sup>14</sup>C]oleate was incorporated into CO<sub>2</sub> and cellular lipid. The highest rate of oleate utilization was observed when both glucose and glutamine were present in the culture medium. The presence of oleate in the culture medium did not affect the rates of utilization of either glucose or glutamine. Of the [<sup>14</sup>C]oleate incorporated into lipid, approx. 80% was incorporated into triacylglycerol and only 18% into phospholipid. 5. The turnover rate for the total ATP content of the macrophage in culture is about 10 times per minute: the value for the perfused isolated maximally working rat heart is 22. This indicates a high metabolic rate for macrophages, and consequently emphasizes the importance of the provision of fuels for their function in an immune response.

L11 ANSWER 20 OF 24 MEDLINE on STN  
ACCESSION NUMBER: 86028521 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 4053296  
TITLE: Studies on the site of the block in gluconeogenesis causing severe hypoglycemia in intestinal ischemia shock in rats.  
AUTHOR: van der Meer C; Valkenburg P W; Snijders P M  
SOURCE: Circulatory shock, (1985) Vol. 16, No. 2, pp. 213-28.  
Journal code: 0414112. ISSN: 0092-6213.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 198512  
ENTRY DATE: Entered STN: 21 Mar 1990  
Last Updated on STN: 3 Feb 1997  
Entered Medline: 9 Dec 1985

AB Intestinal ischemia shock was induced by 35 to 40-min portal vein occlusion (PVO). After treatment with rat plasma a severe hypoglycemia ensues which is caused by a block in gluconeogenesis. This hypoglycemia is not affected by treatment with adrenaline, glucagon, nicotinadenine dinucleotide (NAD), adenosine triphosphate (ATP) alanine (A), or pyruvate (P), while fructose (F) and dihydroxyacetone (DHA) slightly increase the plasma glucose concentration. If F or DHA are combined with NAD a considerable hyperglycemic effect is observed, but NAD plus A or P is ineffective. A similar marked rise in plasma glucose is observed if F is combined with nicotinamide, adenylic acid, or histamine. NAD causes vasodilatation in the splanchnic area and an increased portal flow. It is concluded that the effect of NAD is the result of an increased uptake of suitable substrates of gluconeogenesis from the peritoneal cavity and/or an increased availability of these substrates to the liver. During the development of PVO shock, portal venous flow diminishes considerably. This reduced flow may be the result of vasoconstriction caused by the high level of plasma adrenaline.

L11 ANSWER 21 OF 24 MEDLINE on STN  
ACCESSION NUMBER: 85307048 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 3929783  
TITLE: Leukotriene production in rat peritoneal  
leukocytes requires intact energy metabolism.  
AUTHOR: Ahnfelt-Ronne I; Olsen U B  
SOURCE: Biochemical pharmacology, (1985 Sep 1) Vol. 34, No. 17, pp.  
3095-100.  
Journal code: 0101032. ISSN: 0006-2952.  
PUB. COUNTRY: ENGLAND: United Kingdom  
DOCUMENT TYPE: (IN VITRO)  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 198510  
ENTRY DATE: Entered STN: 20 Mar 1990  
Last Updated on STN: 20 Mar 1990  
Entered Medline: 3 Oct 1985

AB Compounds which inhibit cellular production of ATP either by uncoupling of oxidative phosphorylation (valinomycin, carbonylcyanide-4-trifluoromethoxyphenylhydrazone, and 2,4-dinitrophenol), glycolytic phosphorylation (2-deoxy-D-glucose) or by inhibiting respiratory-chain reactions (antimycin A) were all shown to inhibit calcium-ionophore A23187-induced leukotriene synthesis in rat peritoneal leukocytes at concentrations closely correlating with those needed to block ATP synthesis. In contrast, none of the compounds interfered with cyclo-oxygenase or other enzymes involved in arachidonate metabolism in these cells. Two well-known inhibitors of 5-lipoxygenase, nordihydroguaiaretic acid and phenidone, blocked LTB<sub>4</sub> synthesis without affecting ATP production. In conclusion, rat peritoneal leukocyte leukotriene synthesis depends on intact energy metabolism.

L11 ANSWER 22 OF 24 MEDLINE on STN  
ACCESSION NUMBER: 84037103 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 6195384  
TITLE: Studies on the mechanisms of the inhibitory effect of N-5'  
on histamine release from rat peritoneal exudate  
cells.  
AUTHOR: Kubota T; Ujiie A; Naito J; Nakazawa M; Koda A  
SOURCE: Japanese journal of pharmacology, (1983 Aug) Vol. 33, No.  
4, pp. 837-43.  
Journal code: 2983305R. ISSN: 0021-5198.  
PUB. COUNTRY: Japan  
DOCUMENT TYPE: (IN VITRO)  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 198312  
ENTRY DATE: Entered STN: 19 Mar 1990  
Last Updated on STN: 25 Apr 1995  
Entered Medline: 20 Dec 1983

AB To investigate the mechanisms for the inhibition of IgE-mediated histamine release from rat peritoneal exudate cells (PEC) by N-5', we studied the relation between the inhibitory effect of N-5' on histamine release and the intracellular levels of adenine nucleotides such as ATP and cAMP. Evident histamine release was induced by the addition of specific antigen to rat PEC sensitized with IgE antiserum in vitro, and the release showed a maximum 30 sec after the antigen challenge. In the same time course as the histamine release, the intracellular levels of ATP and cAMP decreased. N-5' significantly inhibited the histamine release and a decrease in ATP level as a result of the antigen-antibody reaction. A decrease in cAMP level showed a tendency to be suppressed by N-5'. Antigen-induced <sup>14</sup>CO<sub>2</sub> production for <sup>6-14</sup>C-glucose in the

sensitized PEC was 3 times that seen in the case without antigen. N-5' dramatically suppressed the acceleration in the production of  $^{14}\text{CO}_2$ . Differing from the action of papaverine, the inhibitory effect of N-5' on the IgE-mediated histamine release from rat PEC was identical both in the presence or in the absence of glucose. N-5' scarcely affected the ATP level in the non-sensitized PEC in the glucose-free medium. On the other hand, N-5' inhibited the activity of  $\text{Na}^+$ ,  $\text{K}^+$ -ATPase, one of the ATP-consuming enzymes, in a dose-dependent fashion. From these results, it is presumed that the suppression of ATP-utilization through the inhibition of  $\text{Na}^+$ ,  $\text{K}^+$ -ATPase activity is involved in the inhibition of histamine release by N-5'. The relation between the inhibitory effect of N-5' on histamine release and both nucleotides was also discussed.

L11 ANSWER 23 OF 24 MEDLINE on STN  
 ACCESSION NUMBER: 76076878 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 172850  
 TITLE: Pyruvate dehydrogenase phosphatase deficiency: a cause of congenital chronic lactic acidosis in infancy.  
 AUTHOR: Robinson B H; Sherwood W G  
 SOURCE: Pediatric research, (1975 Dec) Vol. 9, No. 12, pp. 935-9.  
 Journal code: 0100714. ISSN: 0031-3998.  
 PUB. COUNTRY: Switzerland  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 197602  
 ENTRY DATE: Entered STN: 13 Mar 1990  
 Last Updated on STN: 3 Feb 1997  
 Entered Medline: 13 Feb 1976

AB A male child presented on the first day of life with metabolic acidosis with elevated blood lactate (15 mM), pyruvate (0.4 mM), and free fatty acid (1.3 mM) levels and a blood pH of 7.16. The severity of the acidosis was diminished by intravenous administration of glucose in large doses and by bicarbonate. On two occasions, when the acidosis was particularly severe, peritoneal dialysis using an acetate buffer was required. Restriction of the dietary intake of saturated fatty acids or treatment with nicotinic acid also appeared to diminish the severity of acidosis. No improvement was achieved by the administration of thiamine or biotin. Tissues taken at postmortem showed normal activity of gluconeogenic enzymes and pyruvate dehydrogenase. The activity of pyruvate dehydrogenase in tissue homogenates preincubated with ATP was reduced by 60-75% both in liver of the patient and of the controls because of the inactivation of the enzyme by pyruvate dehydrogenase kinase. Addition of  $\text{Ca}^{++}$  and  $\text{Mg}^{++}$  to the inactivated enzyme caused a prompt return of the activity to normal in controls but not in the patient. This defect, which was apparent in muscle and liver but not in brain, we attribute to a markedly reduced activity of pyruvate dehydrogenase phosphatase in the patient.

L11 ANSWER 24 OF 24 MEDLINE on STN  
 ACCESSION NUMBER: 71087407 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 5312926  
 TITLE: [Metabolic inductions in the rat after an intraperitoneal injection of fructose and glucose, according to the nature of the dietary carbohydrate. I. Modifications after a month of the diet].  
 Inductions metaboliques chez le rat, a la suite d'une injection intra-peritoneale de fructose et de glucose, selon la nature des glucides du regime. I. Modifications apres un mois de regime.  
 AUTHOR: Baron P; Griffaton G; Lowy R  
 SOURCE: Enzymologia biologica et clinica, (1970) Vol. 11, No. 6, pp. 538-54.

PUB. COUNTRY: Switzerland  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: French  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 197103  
ENTRY DATE: Entered STN: 1 Jan 1990  
Last Updated on STN: 1 Jan 1990  
Entered Medline: 9 Mar 1971

ACCESSION NUMBER: 2003:998636 CAPLUS

DOCUMENT NUMBER: 141:111326

TITLE: The in vivo biocompatibility performance of a 25 mmol/L bicarbonate/10 mmol/L lactate-buffered peritoneal dialysis fluid

AUTHOR(S): Skoufos, Line; Topley, Nicholas; Cooker, Laurinda; Dawnay, Anne; Millar, David J.; Holmes, Clifford J.; Faict, Dirk

CORPORATE SOURCE: Renal Division, Baxter Healthcare Corporation, McGaw Park, IL, USA

SOURCE: Kidney International, Supplement (2003), 88, S94-S99  
CODEN: KISUDF; ISSN: 0098-6577

PUBLISHER: Blackwell Science, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The biocompatibility profile of a new peritoneal dialysis (PD) solution (Physioneal 35) was determined using a selection of in vitro assay systems. Physioneal 35 is buffered by a combination of 25 mmol/L bicarbonate and 10 mmol/L lactate, thereby providing a solution with a total of 35 mmol/L of alkali to complement the currently available 25 mmol/L bicarbonate and 15 mmol/L lactate combination solution, Physioneal 40. In addition, the new solution contains a calcium concentration of 1.75 mmol/L

rather than

1.25 mmol/L present in Physioneal 40. Physioneal 35 and 40 are manufactured in double chamber bag systems that permit separation of glucose from the buffer during sterilization. When the two chambers are mixed just before patient use, the resulting solution has a neutral pH and reduced glucose degradation content. Physioneal 35 was evaluated for its cytotoxicity potential using a murine fibroblast assay, its acute effect on human neutrophil and human peritoneal mesothelial cell function, and its in vitro potential to form advanced glycation end products (AGE). The biocompatibility characteristics of this new formulation were compared with that of a conventional, lactate-based solution and to that of its parent formulation, Physioneal 40. Proliferation of murine fibroblasts was determined after exposure to dialysis fluids for 72 h. Cell viability was assayed by the ability to take up neutral red dye. Human neutrophils were exposed for 15 min to dialysis fluids, and their ATP content and phorbol 12-myristate 13-acetate (PMA) stimulated chemiluminescence response was determined as a measure of viability and respiratory burst activity, resp. Cellular interleukin (IL)-1 $\beta$ -driven IL-8 synthesis by human mesothelial cells following acute exposure to dialysis fluids was also assessed. Advanced glycation end product formation in the dialysis fluids was measured after 5 and 20 days of incubation with human serum albumin (HSA) as the model protein. In all assays employed, the biocompatibility profile of Physioneal 35 was similar to that of the Physioneal 40 parent formulation. Physioneal 35 showed a significant improvement in biocompatibility performance compared to a pH neutralized conventional lactate-buffered peritoneal dialysis solution in the murine fibroblast assay. In the acute exposure assays, human neutrophil viability and respiratory burst were significantly improved compared with the acidic, conventional solution; however, no statistically significant improvement were seen in mesothelial cells. AGE formation, which is thought to be an important mechanism by which glucose and glucose degradation products cause structural and functional changes of the peritoneal membrane, was significantly lower in Physioneal 35 compared with the conventional dialysis solution. The biocompatibility profile of Physioneal 35 was similar to that of the original Physioneal 40 bicarbonate/lactate-buffered dialysis solution, confirming that differences in both buffer content and calcium concentration do not affect biocompatibility performance. Both bicarbonate/lactate formulations (Physioneal 35 and Physioneal 40) were more biocompatible than a conventional lactate-buffered dialysis



solution in this in vitro biocompatibility assessment.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 2 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1989:454948 CAPLUS

DOCUMENT NUMBER: 111:54948

TITLE: Rates of utilization of glucose, glutamine and oleate and formation of end-products by mouse peritoneal macrophages in culture

AUTHOR(S): Newsholme, Philip; Newsholme, Eric A.

CORPORATE SOURCE: Dep. Biochem., Univ. Oxford, Oxford, OX1 3QU, UK

SOURCE: Biochemical Journal (1989), 261(1), 211-18

CODEN: BIJOAK; ISSN: 0306-3275

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The metabolism of mouse thioglycollate-elicited peritoneal macrophages was studied in culture for  $\leq 96$  h. The rates of glycolysis, lactate formation, and glutamine (I) utilization were approx. linear with time for  $\geq 80$  h of culture. The rates of glucose (II) and I utilization by cultured macrophages were .apprx.500 nmol/h/mg and .apprx.90 nmol/h/mg protein, resp. This rate of II utilization was  $\geq 50\%$  greater than that previously reported for macrophages during 60-min incubation in a shaking flask; and it was increased by addition of I to the culture medium. The rate of I utilization in culture was similar to that previously reported for macrophages during 60-min incubation. The major end-product of II metabolism was lactate, and those of I metabolism were CO<sub>2</sub>, glutamate, NH<sub>3</sub>, and alanine. <sup>14</sup>C from [<sup>14</sup>C]oleate ([<sup>14</sup>C]III) was incorporated into CO<sub>2</sub> and cellular lipid in these cells. The highest rate of III utilization was observed when both I and II were present in the culture medium. The presence of III in the culture medium did not affect the rates of utilization of either I or II. Of the [<sup>14</sup>C]III incorporated into lipid, .apprx.80% was incorporated into triacylglycerol and only 18% into phospholipid. The turnover rate for the total ATP content of the macrophage in culture was .apprx.10 times per min: the value for the perfused isolated maximally working rat heart is 22. This indicates a high metabolic rate for macrophages and consequently emphasizes the importance of the provision of fuels for their function in an immune response.

L11 ANSWER 3 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1989:405153 CAPLUS

DOCUMENT NUMBER: 111:5153

TITLE: Effect of fluid infusion on alterations in metabolite levels in carbohydrate and protein metabolism of septic rats

AUTHOR(S): Mori, Eigo; Hasebe, Masaharu; Kobayashi, Kunio

CORPORATE SOURCE: Sch. Med., Teikyo Univ., Kaga, 173, Japan

SOURCE: Journal of Clinical Biochemistry and Nutrition (1988), 5(3), 241-54

CODEN: JCBNER; ISSN: 0912-0009

DOCUMENT TYPE: Journal

LANGUAGE: English

AB This study evaluates the effect of continuous fluid infusion on metabolic alterations in septic rats. Saline administration, designed to maintain blood volume, was carried out from the onset of peritonitis induced by cecal ligation and puncture. The operated rats were divided into two groups: the physiol. saline-resuscitated group, treated by continuous infusion i.v. (2.0 mL/h) immediately after surgery, and the untreated septic group. Concns. of hepatic adenine nucleotides, glycogen, glucose 6-phosphate, phosphoenolpyruvate, lactate, malate, RNA, DNA, and protein were determined at 4, 7, 12, and 24 h. Alterations in free amino acid levels in plasma and liver were also measured. A rapid decrease in plasma glucogenic amino acids and perturbation

of carbohydrate metabolism were noted at 4 h and alterations in metabolite levels became evident at 7 h in both septic rat groups, when compared with those in normal rats. In addition, obvious differences in the alteration patterns of metabolite contents were observed at 12 h between the two septic rat groups. The untreated septic rats appeared to be in a state of shock at 12 h and showed .apprx.50% mortality, whereas all resuscitated septic rats were alive at this time. Thus, an adverse effect of lowered tissue perfusion was involved in further aggravation of the deterioration of hepatic ATP-producing metabolism in the untreated septic rats.

L11 ANSWER 4 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1987:632431 CAPLUS

DOCUMENT NUMBER: 107:232431

TITLE: Determination of nucleotides, nucleosides and nucleobases in cells of different complexity by reversed-phase and ion-pair high-performance liquid chromatography

AUTHOR(S): Werner, Andreas; Siems, Werner; Schmidt, Heike; Rapoport, Iris; Gerber, Gerhard; Toguzov, R. T.; Tikhonov, Yu. V.; Pimenov, A. M.

CORPORATE SOURCE: Inst. Biochem., Humboldt Univ. Berlin, Berlin, 1040, Ger. Dem. Rep.

SOURCE: Journal of Chromatography (1987), 421(2), 257-65  
CODEN: JOCRAM; ISSN: 0021-9673

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Procedures are presented for the anal. of profiles of purine and pyridine compds. in human and rabbit red blood cells by reversed-phase HPLC and in Ehrlich ascites tumor cells of mouse by ion-pair HPLC. These compds. are present in rabbit erythrocytes in higher concns. than in human blood cells, and in rabbit reticulocytes the concentration of purine compds. is still higher. During glucose-free incubation, human red cells accumulate adenosine and adenine in the presence of coformycin owing to the inhibition of adenosine and AMP deamination. Ehrlich ascites tumor cells lose major portions of purine mono-, di- and triphosphates between the seventh and eleventh day after inoculation into mouse peritoneal cavities.

L11 ANSWER 5 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1987:16587 CAPLUS

DOCUMENT NUMBER: 106:16587

TITLE: Alterations in metabolite levels in carbohydrate and energy metabolism of rat in hemorrhagic shock and sepsis

AUTHOR(S): Mori, Eigo; Hasebe, Masaharu; Kobayashi, Kunio; Iijima, Norimasa

CORPORATE SOURCE: Sch. Med., Teikyo Univ., Tokyo, 173, Japan

SOURCE: Metabolism, Clinical and Experimental (1987), 36(1), 14-20

CODEN: METAAJ; ISSN: 0026-0495

DOCUMENT TYPE: Journal

LANGUAGE: English

AB For comparison of the extent of metabolite content alteration caused by etiol. different types of shock, septic peritonitis and hemorrhagic shock were produced in rats. Contents of metabolites were determined in the liver and muscles. Characteristic differences were found in the alteration modes of hepatic lactate level, muscle adenine nucleotide concns., and muscle protein content between these shock models. Rapid and significant alterations were observed in the levels of adenine nucleotides, glucose 6-phosphate and lactate in the liver in both types of shock. Hepatic energy charge and contents of glycogen and protein also decreased. Noticeable changes in the muscles were elevation of lactate level and the decrease of phosphocreatine and protein concns. Another distinct change was the decrease of total adenine nucleotide content in

the muscle of septic rats, whereas it remained unchanged in the muscle of hemorrhagic shock rats. Thus, the changes of metabolite levels did not occur simultaneously in different tissues, and their rate and magnitude varied between different types of shock. The difference in adaptive response of metabolism may result in pathophysiol. diversity in shock.

L11 ANSWER 6 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1985:554801 CAPLUS

DOCUMENT NUMBER: 103:154801

TITLE: Leukotriene production in rat peritoneal leukocytes requires intact energy metabolism

AUTHOR(S): Ahnfelt-Roenne, Ian; Olsen, Uffe Bang

CORPORATE SOURCE: Dep. Pharmacol., Leo Pharm. Prod., Ballerup, DK-2750, Den.

SOURCE: Biochemical Pharmacology (1985), 34(17), 3095-100  
CODEN: BCPA6; ISSN: 0006-2952

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Compds. which inhibit cellular production of ATP [56-65-5] by uncoupling of oxidative phosphorylation (valinomycin, carbonylcyanide-4-trifluoromethoxyphenylhydrazine, and 2,4-dinitrophenol), by affecting glycolytic phosphorylation (2-deoxy-D-glucose), or by inhibiting respiratory-chain reactions (antimycin A) were all shown to inhibit calcium-ionophore A23187-induced leukotriene (LTB<sub>4</sub> [71160-24-2]) synthesis in rat peritoneal leukocytes at concns. closely correlating with those needed to block ATP synthesis. In contrast, none of the compds. interfered with cyclooxygenase or other enzymes involved in arachidonate metabolism in these cells. Two well-known inhibitors of 5-lipoxygenase, nordihydroguaiaretic acid and phenidone, blocked LTB<sub>4</sub> synthesis without affecting ATP production. In conclusion, rat peritoneal leukocyte leukotriene synthesis depends on intact energy metabolism

L11 ANSWER 7 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1985:469666 CAPLUS

DOCUMENT NUMBER: 103:69666

TITLE: Role of 2-deoxy-D-glucose in the inhibition of phagocytosis by mouse peritoneal macrophage

AUTHOR(S): Sung, Sun Sang J.; Silverstein, Samuel C.

CORPORATE SOURCE: Lab. Cell. Physiol. Immunol., Rockefeller Univ., New York, NY, 10021, USA

SOURCE: Biochimica et Biophysica Acta, Molecular Cell Research (1985), 845(2), 204-15  
CODEN: BBAMCO; ISSN: 0167-4889

DOCUMENT TYPE: Journal

LANGUAGE: English

AB 2-Deoxy-D-glucose (I) inhibits Fc and complement receptor-mediated phagocytosis of mouse peritoneal macrophages. To understand the mechanism of this inhibition, I metabolites were analyzed in macrophages under phagocytosis inhibition conditions and conditions of phagocytosis reversal caused by glucose, mannose and 5-thio-D-glucose, and their accumulations were compared under these conditions. Macrophages metabolized I to form 2-deoxy-D-glucose 6-phosphate, 2-deoxy-D-glucose 1-phosphate, UDP-2-deoxy-D-glucose, 2-deoxy-D-glucose 1,6-diphosphate, 2-deoxy-D-gluconic acid and 2-deoxy-6-phospho-D-gluconic acid. The level of bulk accumulation as well as the accumulation of any of these I metabolites did not correlate with changes in macrophage phagocytosis capacities caused by the reversing sugars. I inhibited glycosylation of thioglycolate-elicited macrophage by 70-80%. This inhibition did not cause phagocytosis inhibition. The inhibition of protein synthesis by I similarly could not account for phagocytosis inhibition, since cycloheximide, when used at a

concentration that inhibited protein synthesis by 95%, did not affect phagocytosis. I lowered cellular nucleoside triphosphates by 70-99%, but their intracellular levels in the presence of different reversing sugars did not correlate with the magnitude of phagocytosis reversal caused by the sugars. Thus, I inhibits phagocytosis by a mechanism distinct from its usual action of inhibiting glycosylation, protein synthesis and depleting energy supplies, mechanisms by which I inhibits other cellular processes.

L11 ANSWER 8 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1979:521152 CAPLUS  
DOCUMENT NUMBER: 91:121152  
TITLE: Comparative metabolic studies on liver sinusoidal cells and different types of macrophages  
AUTHOR(S): Hofmann, Friedrich; Decker, Karl  
CORPORATE SOURCE: Med. Fak., Univ. Freiburg, Freiburg, D-7800, Fed. Rep. Ger.  
SOURCE: Hoppe-Seyler's Zeitschrift fuer Physiologische Chemie (1979), 360(7), 905-12  
CODEN: HSZPAZ; ISSN: 0018-4888  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Sinusoidal cells of rat liver, rat peritoneal macrophages, and rabbit alveolar macrophages were studied for their viability and compared with regard to cell weight and protein and DNA contents. Glycogen was virtually absent from sinusoidal cells and peritoneal macrophages; alveolar macrophages contained glycogen, the level of which increased after activation by Freund's adjuvant and decreased during phagocytosis in the absence of glucose. .: Of the different nucleotides assayed, UDP-glucose levels were low in nonglycogen-forming cells, but quite high in alveolar macrophages. The capacity to metabolize galactose was much less in all cell types investigated than in hepatocytes.

L11 ANSWER 9 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1978:421081 CAPLUS  
DOCUMENT NUMBER: 89:21081  
TITLE: Nucleotide contents and nitrogen metabolism of rat Kupffer cells and peritoneal macrophages  
AUTHOR(S): Decker, Karl; Hofmann, Friedrich; Kreusch, Juergen; Maier, Klaus P.; Munder, Paul G.; Wagle, Shreepad R.  
CORPORATE SOURCE: Biochem. Inst., Albert-Ludwigs-Univ., Freiburg/Br., Fed. Rep. Ger.  
SOURCE: Kupffer Cells Other Liver Sinusoidal Cells, Proc. Int. Kupffer Cell Symp. (1977), 315-24. Editor(s): Wisse, E.; Knook, D. L. Elsevier: Amsterdam, Neth.  
CODEN: 38EFAW  
DOCUMENT TYPE: Conference  
LANGUAGE: English

AB Kupffer cells prepared simultaneously with hepatocytes by an improved procedure were compared quant. with peritoneal macrophages. The weak response to D-galactosamine could be attributed to a small capacity of the galactose pathway. Both cell types were able to take up and phosphorylate uridine. Among the nucleotide contents determined, the very low levels of UDPG and UDP-galactose, as compared to hepatocytes, were remarkable. The ability to produce urea from amino acids was similar in Kupffer cells and in hepatocytes; peritoneal macrophages also synthesized urea, although on a much smaller scale. The presence of all enzymes of the urea cycle could be demonstrated; the activity of argininosuccinate synthetase corresponded with the overall rate of urea formation by the resp. intact cells.

L11 ANSWER 10 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1968:47703 CAPLUS

DOCUMENT NUMBER: 68:47703  
TITLE: Histamine release from isolated rat peritoneal mast cells induced by adenosine 5'-triphosphate  
AUTHOR(S): Diamant, Bertil; Kruger, Per G.  
CORPORATE SOURCE: Karolinska Inst., Stockholm, Swed.  
SOURCE: Acta Physiologica Scandinavica (1967), 71(4), 291-302  
CODEN: APSCAX; ISSN: 0001-6772  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Histamine release from isolated rat mast cells is induced by ATP ( $10^{-5}$  -  $3 \times 10^{-5}$ M). The activity found in an ADP preparation was shown to be due to contamination with ATP. Purified ADP has no activity. AMP, 3'-AMP, phosphocreatine, and 2-phosphoenolpyruvic acid were without histamine-releasing activity. Histamine release induced by ATP was compared with that caused by compound 48/80 with respect to the influence of  $Ca^{++}$ ,  $Mg^{++}$ ,  $Zn^{++}$ , ouabain, 2,4-dinitrophenol, oligomycin, and glucose. The results are interpreted to indicate that ATP and compound 48/80 release histamine by mechanisms that in certain respects differ from each other. The histamine-releasing effect of ATP is discussed in relation to the energy-requiring mechanism that is known to be involved in histamine release by compound 48/80.

L11 ANSWER 11 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1962:465876 CAPLUS  
DOCUMENT NUMBER: 57:65876  
ORIGINAL REFERENCE NO.: 57:13131e-f  
TITLE: Antidiabetic biguanides  
AUTHOR(S): Ditschuneit, H.; Lotz, W.; Fritzsche, W.; Pfeiffer, E. F.  
CORPORATE SOURCE: Univ. Klin., Frankfurt, Germany  
SOURCE: Congr. Federation Intern. Diabète, 4, Geneva, Switz. (1961), (1), 740-3  
DOCUMENT TYPE: Journal  
LANGUAGE: Unavailable

AB Glucose utilization and O consumption in isolated rat adipose tissue and peritoneum are increased in the presence of N1-phenylethylbiguanide (DBI). DBI injections in rats cause increased levels of fructose 1,6-diphosphate and lactic acid in the liver, but decreases of adenosine triphosphate and elimination of methemoglobin reduction

L16 ANSWER 1 OF 2 MEDLINE on STN  
 ACCESSION NUMBER: 2003125495 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 12640175  
 TITLE: Prolonged neonatal seizures exacerbate hypoxic-ischemic brain damage: correlation with cerebral energy metabolism and excitatory amino acid release.  
 AUTHOR: Yager Jerome Y; Armstrong Edward A; Miyashita Hero; Wirrell Elaine C  
 CORPORATE SOURCE: Department of Pediatrics, University of Saskatchewan, Saskatoon, Canada.. yager@duke.usask.ca  
 SOURCE: Developmental neuroscience, (2002) Vol. 24, No. 5, pp. 367-81.  
 Journal code: 7809375. ISSN: 0378-5866.  
 PUB. COUNTRY: Switzerland  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200305  
 ENTRY DATE: Entered STN: 18 Mar 2003  
 Last Updated on STN: 9 May 2003  
 Entered Medline: 8 May 2003

AB BACKGROUND: Perinatal hypoxia-ischemia (HI) is the most common precipitant of seizures in the first 24-48 h of a newborn's life. In a previous study, our laboratory developed a model of prolonged, continuous electrographic seizures in 10-day-old rat pups using kainic acid (KA) as a proconvulsant. Groups of animals included those receiving only KA, or HI for 15 or 30 min, followed by KA infusion. Our results showed that prolonged electrographic seizures following 30 min of HI resulted in a marked exacerbation of brain damage. We have undertaken studies to determine alterations in hippocampal high-energy phosphate reserves and the extracellular release of hippocampal amino acids in an attempt to ascertain the underlying mechanisms responsible for the damage promoted by the combination of HI and KA seizures. METHODS: All studies were performed on 10-day-old rats. Five groups were identified: (1) group I--KA alone, (2) group II--15 min of HI plus KA, (3) group III--15 min of HI alone, (4) group IV--30 min of HI plus KA, and (5) group VI--30 min of HI alone. HI was induced by right common carotid artery ligation and exposure to 8% oxygen/balance nitrogen. Glycolytic intermediates and high-energy phosphates were measured. Prior to treatment, at the end of HI (both 15 and 30 min), prior to KA injection, and at 1 (onset of seizures), 3, 5 (end of seizures), 7, 24 and 48 h, blood samples were taken for glucose, lactate and beta-hydroxybutyrate. At the same time points, animals were sacrificed by decapitation and brains were rapidly frozen for subsequent dissection of the hippocampus and measurement of glucose, lactate, beta-hydroxybutyrate, adenosine triphosphate (ATP) and phosphocreatine (PCr). In separate groups of rats as defined above, microdialysis probes (CMA) were stereotactically implanted into the CA2-3 region of the ipsilateral hippocampus for measurement of extracellular amino acid release. Dialysate was collected prior to any treatment, at the end of HI (15 and 30 min), prior to KA injection, and at 1 (onset of seizures), 3, 5 (end of seizures), 7 and 9 h. Determination of glutamate, serine, glutamine, glycine, taurine, alanine, and GABA was accomplished using high-performance liquid chromatography with EC detection. RESULTS: Blood and hippocampal glucose concentrations in all groups receiving KA were significantly lower than control during seizures ( $p < 0.05$ ). beta-Hydroxybutyrate values displayed the inverse, in that values were significantly higher ( $p < 0.01$ ) in all KA groups compared with pretreatment controls during seizure activity. Values returned to control by 2 h following the cessation of seizures. Lactate concentrations in brain and blood mimicked those of beta-hydroxybutyrate. ATP values declined to 0.36 mmol/l in both the 15 and 30 min hypoxia groups compared

with 1.85 mmol/l for controls ( $p < 0.01$ ). During seizures, ATP and PCr values declined significantly below their homologous controls. Following seizures, ATP values only for those animals receiving KA plus HI for 30 min remained below their homologous controls for at least 24 h. Determination of amino acid release revealed elevations of glutamate, glycine, taurine, alanine and GABA above pretreatment control during HI, with a return to normal prior to KA injections. During seizures and for the 4 h of recovery monitored, only glutamate in the combined HI and KA group rose significantly above both the 15 min of HI plus KA and the KA alone group ( $p < 0.05$ ). CONCLUSION: Under circumstances in which there is a protracted depletion of high-energy phosphate reserves, as occurs with a combination of HI- and KA-induced seizures, excess amounts of glutamate become toxic to the brain. The latter may account for the exacerbation of damage to the newborn hippocampus, and serve as a target for future therapeutic intervention.

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L16 ANSWER 2 OF 2 MEDLINE on STN  
 ACCESSION NUMBER: 95256469 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 7738204  
 TITLE: Glucose modulates rat substantia nigra GABA release in vivo via ATP-sensitive potassium channels.  
 AUTHOR: During M J; Leone P; Davis K E; Kerr D; Sherwin R S  
 CORPORATE SOURCE: Molecular Pharmacology and Neurogenetics Laboratory, Yale University School of Medicine, New Haven, Connecticut 06520-8039, USA.  
 CONTRACT NUMBER: DK-20495 (NIDDK)  
 NS-06208 (NINDS)  
 NS-28227 (NINDS)  
 SOURCE: The Journal of clinical investigation, (1995 May) Vol. 95, No. 5, pp. 2403-8.  
 Journal code: 7802877. ISSN: 0021-9738.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: (COMPARATIVE STUDY)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
 LANGUAGE: English  
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
 ENTRY MONTH: 199506  
 ENTRY DATE: Entered STN: 15 Jun 1995  
 Last Updated on STN: 15 Jun 1995  
 Entered Medline: 5 Jun 1995  
 AB Glucose modulates beta cell insulin secretion via effects on ATP-sensitive potassium (KATP) channels. To test the hypothesis that glucose exerts a similar effect on neuronal function, local glucose availability was varied in awake rats using microdialysis in the substantia nigra, the brain region with the highest density of KATP channels. 10 mM glucose perfusion increased GABA release by 111 +/- 42%, whereas the sulfonylurea, glipizide, increased GABA release by 84 +/- 20%. In contrast, perfusion of the KATP channel activator, lemakalim, or depletion of ATP by perfusion of 2-deoxyglucose with oligomycin inhibited GABA release by 44 +/- 8 and 45 +/- 11%, respectively. Moreover, the inhibition of GABA release by 2-deoxyglucose and oligomycin was blocked by glipizide. During systemic insulin-induced hypoglycemia (1.8 +/- 0.3 mM), nigral dialysate GABA concentrations decreased by 49 +/- 4% whereas levels of dopamine in striatal dialysates increased by 119 +/- 18%. We conclude that both local and systemic glucose availability influences nigral GABA release via an effect on KATP channels and that inhibition of GABA release may in part mediate the hyperexcitability associated with hypoglycemia. These data support the hypothesis that glucose acts as a signaling molecule, and not simply as an energy-yielding fuel, for neurons.

L17 ANSWER 1 OF 1 MEDLINE on STN  
 ACCESSION NUMBER: 88210904 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 3365863  
 TITLE: Effects of acetate on glucose metabolism and cellular ATP content during hemodialysis.  
 AUTHOR: Panzetta G; Tessitore N; Schiavon R; Panebianco R; Maschio G  
 CORPORATE SOURCE: Division of Nephrology, University of Verona, Italy.  
 SOURCE: Clinical nephrology, (1988 Apr) Vol. 29, No. 4, pp. 179-84. Journal code: 0364441. ISSN: 0301-0430.  
 PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 198806  
 ENTRY DATE: Entered STN: 8 Mar 1990  
 Last Updated on STN: 8 Mar 1990  
 Entered Medline: 13 Jun 1988

AB In view of multiple interactions of acetate with intermediary metabolism, we studied the effects of the exogenous acetate load during dialysis on glucose and energy metabolism. IV glucose tolerance test (glucose 0.33 g/kg BW) and platelet ATP content were evaluated in 16 patients before and after a single hemodialysis session with acetate 38 mEq/l in the dialysate. IV glucose tolerance was greatly impaired in all patients after hemodialysis ( $K: 1.08 \pm 0.30$  vs predialysis value of  $2.05 \pm 0.85$ ,  $p$  less than 0.001). Platelet ATP content was unchanged by dialysis ( $3.74 \pm 1.02$   $\mu\text{mol}/10(11)$  PLTs before and  $3.55 \pm 0.69$   $\mu\text{mol}/10(11)$  PLTs after), however, individual variations in platelet ATP content ranged from +32 to -31% of the initial values. Postdialysis plasma acetate levels ranged from 1.5 to 9.2 mmol/l and were inversely correlated with postdialysis glucose tolerance test ( $r: -0.61$ ,  $p$  less than 0.01) and platelet ATP content variations ( $r: -0.51$ ,  $p$  less than 0.05). Our study demonstrates that glucose utilization is acutely impaired by acetate dialysis and suggests that the reduced glycolytic activity may be due to a negative feed-back mechanism in the presence of exogenous fuel. It also demonstrates a great variability in platelet ATP content following hemodialysis, which probably depends on the different patients' ability to oxidize acetate.



L18 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1997:20190 CAPLUS

DOCUMENT NUMBER: 126:99003

TITLE: The effect of aspirin on red cell electrolyte, ATP levels and glucose utilization in rats

AUTHOR(S): Bekpinar, S.; Ozant, F.; Oz, G.

CORPORATE SOURCE: Department of Biochemistry, Istanbul Medical Faculty, University of Istanbul, Istanbul, 34390, Turk.

SOURCE: Pharmaceutical Sciences (1996), 2(7), 345-347  
CODEN: PHSCFB; ISSN: 1356-6881

PUBLISHER: Royal Pharmaceutical Society of Great Britain

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Wistar rats were fed chow supplemented with 1% (weight/weight) aspirin for one month. At 3, 7, 14, 21 and 30 days of treatment, heparinized blood was collected. ATP, Na<sup>+</sup>, K<sup>+</sup> levels, glucose utilization of erythrocytes and inorg. phosphate, Na<sup>+</sup>, K<sup>+</sup>, salicylate levels of plasma were determined. Erythrocyte ATP and plasma phosphate levels were decreased following aspirin treatment. ATP decrement caused erythrocyte K<sup>+</sup> level to diminish significantly. Plasma Na<sup>+</sup> and K<sup>+</sup> levels were not effected by aspirin treatment. In addition, the decrease in ATP levels induced erythrocyte glucose utilization. It was concluded that the therapeutic dose of aspirin in man, gave rise to a decrease in the energy production and an impairment of energy utilizing events in erythrocytes in rats (the therapeutic serum aspirin concentration in man is 25-40 mg dL<sup>-1</sup>).

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1985:571728 CAPLUS

DOCUMENT NUMBER: 103:171728

TITLE: Effects of adenosine triphosphate-magnesium chloride (ATP-magnesium chloride) on shocked red blood cells

AUTHOR(S): Shimizu, Kazuhiro; Kawazoe, Shouichi; Yamada, Osamu; Nagase, Hideo

CORPORATE SOURCE: Res. Dev. Dep., Fuso Pharm. Ind. Ltd., Osaka, 536, Japan

SOURCE: Yakugaku Zasshi (1985), 105(8), 784-90  
CODEN: YKKZAJ; ISSN: 0031-6903

DOCUMENT TYPE: Journal

LANGUAGE: Japanese

AB The effects of ATP-MgCl<sub>2</sub> [1476-84-2] on the membrane electrolytes transport and deformability of shocked red blood cells (RBC) with impaired cellular energy metabolism were studied. Rabbit RBC suspension in phosphate buffered saline was incubated at 37° for 14 h. Cellular ATP [56-65-5] level was reduced to approx. 6% of normal value by this incubation. ATP-MgCl<sub>2</sub> (100 μM), added to the incubation medium along with glucose [50-99-7] (10 mM) after 14 h of incubation, significantly prevented further decreases in cellular ATP and 2,3-diphosphoglycerate [138-81-8] levels. Increases in intracellular Na<sup>+</sup> concentration and K<sup>+</sup> leakage from RBC were counteracted by simultaneous

ATP-MgCl<sub>2</sub> and glucose treatment and pH of the incubation medium showed significant reduction compared with the control. Glucose utility and deformability of shocked RBC were improved by ATP-MgCl<sub>2</sub> and glucose treatment. ATP-MgCl<sub>2</sub> appears to be helpful to restore the impaired RBC function by improving the intracellular energy metabolism

L18 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1984:605525 CAPLUS

DOCUMENT NUMBER: 101:205525

TITLE: Calcium-dependent effects of cadmium on energy metabolism and function of perfused rat heart

AUTHOR(S): Prentice, Robert C.; Hawley, Philip L.; Glonek, Thomas; Kopp, Stephen J.  
CORPORATE SOURCE: Nucl. Magnetic Resonance Lab., Chicago Coll. Osteop. Med., Chicago, IL, 60615, USA  
SOURCE: Toxicology and Applied Pharmacology (1984), 75(2), 198-210  
CODEN: TXAPA9; ISSN: 0041-008X  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Postequilibrated isolated rat hearts were perfused for 60 min with a standard supporting electrolyte buffer containing 1 of the following Ca concns.: 0.9, 1.8, 3.5, or 5.0 mM, either with or without added Cd. Doses of Cd which proved to be minimally (0.03  $\mu$ M Cd) and maximally (3.0  $\mu$ M Cd) effective at 0.9 mM Ca were studied at all other Ca concns. A dose-dependent pos. inotropy that persisted throughout the 60-min perfusion period was induced by the graded increases in the perfusate Ca concentration throughout the range 0.9-5.0 mM. Atrioventricular node conductivity was prolonged significantly in hearts perfused with 0.9 mM Ca as compared to hearts perfused with higher Ca concns. Increasing the perfusate Ca concentration caused a dose-dependent increase in heart glycerol 3-phosphorylcholine (GPC) [563-24-6] content. The other measured phosphatic metabolites of the heart were not altered significantly by varying the perfusate Ca level. In contrast, Cd (3.0  $\mu$ M Cd) induced extensive functional and metabolic aberrations which varied in magnitude as an inverse function of the perfusate Ca concentration. Contractile tension, rate of tension development (dT/dt), heart rate, coronary flow rate, and atrioventricular node conductivity were decreased significantly in response to Cd perfusion. Moreover, these hearts characteristically had significantly elevated low energy phosphate (inosine monophosphate [131-99-7] and inorg. phosphate) and decreased high energy phosphate (ATP [56-65-5] and phosphocreatine [67-07-2]) levels relative to their resp. Ca controls. Furthermore, various phosphorylated intermediates of glycolysis (glucose 6-phosphate [56-73-5], fructose 6-phosphate [643-13-0], glucose 1-phosphate [59-56-3]), as well as glycerol 3-phosphate [57-03-4], and uridine diphosphoglucose [133-89-1] accumulated significantly in hearts perfused with Cd at certain Ca concns. <5.0 mM. The Ca-activated increase in heart GPC was inhibited completely by 3  $\mu$ M Cd. At the minimally ED of Cd (0.03  $\mu$ M), demonstrable changes were apparent only at the lowest perfusate Ca concentration examined (0.9 mM). These findings are consistent with the hypothesis that Cd interferes with Ca-activated and Ca-mediated physiol. and biochem. processes of the mammalian heart. The primary mechanistic basis for the action of Cd appears to be linked to a competition with Ca for membrane and possibly intracellular binding and activation sites.

L18 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1981:202564 CAPLUS  
DOCUMENT NUMBER: 94:202564  
TITLE: Effects of single therapeutic dose of glycerol on cerebral metabolism in the brains of young mice: possible increase in brain glucose transport and glucose utilization  
AUTHOR(S): Thurston, Jean Holowach; Hauhart, Richard E.; Dirgo, John A.  
CORPORATE SOURCE: Sch. Med., Washington Univ., St. Louis, MO, USA  
SOURCE: Journal of Neurochemistry (1981), 36(3), 830-8  
CODEN: JONRA9; ISSN: 0022-3042  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The effects of a single therapeutic dose of glycerol [56-81-5] (2 g/kg,

i.p.) on brain carbohydrate and energy metabolism were studied in normal nursing weanling mice. Findings were correlated with brain water and electrolyte content and metabolite changes in plasma, red blood cells, and liver. Plasma glycerol levels peaked at 21 mM 7.5 min after injection and returned to the control value, 0.16 mM, by 2 h. Plasma Na<sup>+</sup> concentration decreased and plasma protein increased for as long as 2 h after injection. Although red blood cells were freely permeable to glycerol, there was no evidence for glycerol metabolism in these cells. Glycerol levels in liver paralleled those in plasma. Glycerol increased liver glucose [50-99-7] concentration 23% and doubled hepatic glycerol-1-phosphate [57-03-4] levels. Liver ATP [56-65-5] levels were reduced 24%. Brain water concentration was reduced from 7.5 min to 30 min after glycerol injection; brain Na<sup>+</sup> and K<sup>+</sup> levels were unchanged. There was no evidence for glycerol entry into brain. While plasma glucose increased 33%, brain glucose increased 87%. Concomitantly there were statistically increases in fructose-1,6-diphosphate [488-69-7], lactate [50-21-5],  $\alpha$ -ketoglutarate [328-50-7], and malate [6915-15-7] levels. The disproportionately high brain glucose value suggests increased transport of glucose from the blood to the brain. Increases in fructose-1,6-diphosphate, lactate,  $\alpha$ -ketoglutarate, and malate are compatible with an increased metabolic flux in the glycolytic pathway and Krebs citric acid cycle. These changes may result from the effects of the hyperosmolar glycerol solution on the blood-brain barrier and on cerebral glucose utilization. The sustained lowering of plasma Na<sup>+</sup> concentration after glycerol injection suggests a need for monitoring plasma Na<sup>+</sup> levels in the clin. situation. Possible lowering of hepatic ATP levels by the use of glycerol in humans is another concern.

L18 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1979:66702 CAPLUS

DOCUMENT NUMBER: 90:66702

TITLE: Effect of ex vivo perfusion of isolated canine stomach with fluorocarbon on the composition of gastric tissues

AUTHOR(S): Kowalewski, K.; Kolodej, A.

CORPORATE SOURCE: Surg.-Med. Res. Inst., Univ. Alberta, Edmonton, AB, Can.

SOURCE: European Surgical Research (1978), 10(5), 322-8  
CODEN: EUSRBM; ISSN: 0014-312X

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Perfusion of isolated canine stomach with fluorocarbon emulsion (FC-47 [311-89-7] suspended in a mixture of electrolytes plus glucose) for 6 h significantly decreased the contents of ATP [56-65-5], ADP [58-64-0], and AMP [61-19-8] in the gastric tissue, but increased creatine phosphate [67-07-2] content. The water and Na<sup>+</sup> contents were increased, but K<sup>+</sup> concentration was reduced. These results are discussed in terms of organ preservation and energy metabolism

L18 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1974:421134 CAPLUS

DOCUMENT NUMBER: 81:21134

TITLE: Digitalis and electrolytes. Mechanism of potassium magnesium aspartate action

AUTHOR(S): Gross, W.; Hagel, H.; Hein, R.; Maiwald, L.

CORPORATE SOURCE: Med. Poliklin., Univ. Wuerzburg, Wuerzburg, Fed. Rep. Ger.

SOURCE: Fortschritte der Medizin (1973), 91(32), 1281-3, 1285  
CODEN: FMDZAR; ISSN: 0015-8178

DOCUMENT TYPE: Journal

LANGUAGE: German

AB Noncardiac patients were injected with 500 mg K DL-aspartate [14434-35-6]

and 500 mg Mg DL-aspartate, and heparinized blood samples taken before and 2 hr after injection, were analyzed for K [7440-09-7], Na [7440-23-5], ATP [56-65-5], and glucose [50-99-7] immediately, after 3 days of storage at 4.deg., and after 4 and 8 hr at 37.deg.. The injection increased the concentration of K in the plasma and decreased the concns. of erythrocyte K, plasma ATP, and plasma glucose relative to controls. During the initial 4 hr at 37.deg., there was a partial reversal of these changes. Oral doses of either 4 g K citrate [7778-49-6], 8 g KCl, 1.05 g essential phospholipids or 1.05 g K-Mg aspartate during 3 hr was followed by sampling of venous blood 10 hr later. The first 2 salts increased the concentration of K in serum and erythrocytes whereas the K-Mg aspartate and phospholipids decreased the concentration of K<sup>+</sup> in serum and increased its concentration in erythrocytes.

K-Mg

aspartate alone increased the concentration of glucose in serum and decreased its concentration in erythrocytes. These electrolyte shifts produced by K-Mg aspartate were supportive to digitalis therapy.

ACCESSION NUMBER: 1986:510068 CAPLUS

DOCUMENT NUMBER: 105:110068

TITLE: Inhibition of potassium uptake and regulation of membrane-associated magnesium ATPase activity of pea roots by aluminum

AUTHOR(S): Matsumoto, Hideaki; Yamaya, Tomoyuki

CORPORATE SOURCE: Inst. Agric. Biol. Sci., Okayama Univ., Kurashiki, 710, Japan

SOURCE: Soil Science and Plant Nutrition (Tokyo, Japan) (1986), 32(2), 179-88

CODEN: SSPNAW; ISSN: 0038-0768

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The uptake of K<sup>+</sup> by pea was decreased markedly by treatment with AlCl<sub>3</sub>. The decrease was alleviated by co-treatment with Ca<sup>2+</sup>. A plasma membrane-enriched fraction was isolated from pea roots, and the associated ATPase [9000-83-3] was characterized. The activity was highly dependent on the presence of Mg<sup>2+</sup>, but not Ca<sup>2+</sup>, and the optimum activity was observed at neutral pH. The activity was inhibited by diethylstilbesterol, DCCD, and vanadate. A rather specific requirement for ATP [56-65-5] was observed. The membrane-associated ATPase was competitively inhibited by AlCl<sub>3</sub> added to the assay medium with respect to ATP. The rate of inhibition by Al at various pHs was parallel with that of the activity in the absence of Al. Various chemical were tested for the alleviation of the inhibition of membrane-associated ATPase by Al. Tripolyphosphate, citric acid [77-92-9], glucose 6-phosphate [56-73-5], pyrophosphate, glutamic acid [56-86-0], and malic acid [6915-15-7] restored the activity in this order. The activity of membrane-associated ATPase treated with Al in vitro was not changes when the unbound Al was removed by dialysis. In contrast the activity of the membrane-associated ATPase prepared from the roots treated with Al in vivo increased. Furthermore, alkaline phosphatase (p-nitrophenyl phosphatase [9001-78-9]) in pea roots increased after the treatment with Al.

L21 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:867880 CAPLUS

DOCUMENT NUMBER: 140:15635

TITLE: Influence of glucose in dialyzing fluid on purine concentrations in hemodialyzed patients with chronic renal failure

AUTHOR(S): Bober, Joanna; Kedzierska, Karolina; Safranow, Krzysztof; Kwiatkowska, Ewa; Jakubowska, Katarzyna; Herdzik, Edyta; Dolegowska, Barbara; Domanski, Leszek; Ciechanowski, Kazimierz

CORPORATE SOURCE: Department of Biochemistry and Chemistry, Pomeranian Medical University, Szczecin, Pol.

SOURCE: Nephron (2003), 95(1), c31-c36

CODEN: NPRNAY; ISSN: 0028-2766

PUBLISHER: S. Karger AG

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In chronic renal failure the accumulation of some purine nucleotides (in erythrocytes) develops both in patients undergoing conservative treatment and in hemodialyzed patients. The aim of the study was: (1) To find if hemodialysis (HD) sessions using dialyzing fluid containing glucose leads to an increase in ATP concentration and changes in the concentration of other nucleotides, nucleosides and oxypurines in erythrocytes. The potential consequence of such purine concentration changes is the increase of 2,3-DPG concentration and an

improved transportation of oxygen in erythrocytes which are more resistant to hemolysis. (2) To compare blood concns. of purine nucleotides, nucleosides and oxypurines in patients undergoing chronic HD with dialyzing fluid containing or lacking glucose. Significant differences could suggest the long-term influence of glucose in dialyzing fluid on erythrocyte energetic state. Whole blood nucleotide concns. were evaluated with the use of a HPLC technique. Before the HD session the patients in the 'plus glucose' group had significantly higher concns. of ATP, ADP, AMP, TAN, NAD, NADP, GTP + GDP, GMP, Urd and HYP than patients in the 'no glucose' group. After the HD the patients in the 'plus glucose' group had significantly higher concns. of ADP, AMP, TAN, NAD, NADP, Urd and HYP than in the 'no glucose' group. Both before and after the HD session, the uric acid concns. and, AEC were significantly lower in the 'plus glucose' group than in the 'no glucose' group. A significant decrease in the whole blood hypoxanthine ( $p < 0.05$ ) and uric acid ( $p < 0.001$ ) concns. after HD was found in the 'no glucose' group while a significant increase in ADP concentration ( $p < 0.05$ ) was detected in the patients' erythrocytes in the 'plus glucose' group. In this group a significant decrease of GTP + GDP and GMP ( $p < 0.05$ ), uric acid concentration

(p  $< 0.001$ ) and adenylate energy charge ( $p < 0.05$ ) were observed after the dialysis. However, no significant differences in nucleotide concns. before and after the HD were found in the 'no glucose' group. Conclusion: The presence of glucose in the dialyzing fluid causes a significant modification of the energetic state of cells which is reflected by the purines' and their metabolites' concns. in the erythrocytes. Higher ATP concns. in patients with renal failure who have been dialyzed with the fluid containing glucose can be considered as an organism adaptation to a decreased amount of RBC and Hb concentration

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1986:219336 CAPLUS

DOCUMENT NUMBER: 104:219336

TITLE: Effect of glucose, NADH and NADPH on cortisol metabolism by mononuclear cells

AUTHOR(S): Klein, A.; Chan, A. W. L.; Malkin, A.  
CORPORATE SOURCE: Sunnybrook Med. Cent., Univ. Toronto, Toronto, ON, M4N  
3M5, Can.  
SOURCE: Journal of Endocrinology (1986), 109(2), 181-5  
CODEN: JOENAK; ISSN: 0022-0795  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Mononuclear cell preps. are capable of metabolizing cortisol [50-23-7] to 3 metabolites which lack the immunosuppressive effect of their precursor. A linear correlation, up to a point, was noted between glucose [50-99-7] concentration and the rate of human mononuclear cell cortisol metabolism in vitro. The mechanism by which glucose exerts its effect was investigated further. The effect of glucose on mononuclear cell cortisol metabolism was not influenced by insulin, and NADPH [53-57-6] and NADH [58-68-4] enhanced cortisol metabolism by disrupted cells, irresp. of whether the homogenates were dialyzed or not. Lactate [50-21-5] and 5'-ATP [56-65-5] inhibited mononuclear cell cortisol metabolism and almost all the glucose used was converted to lactate. Evidently, mononuclear cell cortisol metabolism depends on both nucleotides.

L23 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:453073 CAPLUS  
DOCUMENT NUMBER: 140:429098  
TITLE: Peritoneal dialysis method with solution containing ATP  
INVENTOR(S): Kiribayashi, Kei; Yorioka, Noriaki  
PATENT ASSIGNEE(S): Kowa Co., Ltd., Japan  
SOURCE: PCT Int. Appl., 21 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: Japanese  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004045679	A1	20040603	WO 2003-JP14790	20031120
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
CA 2506704	A1	20040603	CA 2003-2506704	20031120
AU 2003284594	A1	20040615	AU 2003-284594	20031120
EP 1563858	A1	20050817	EP 2003-774083	20031120
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK			
CN 1713926	A	20051228	CN 2003-80103652	20031120
NZ 539796	A	20061130	NZ 2003-539796	20031120
US 2006019925	A1	20060126	US 2005-533538	20050502
PRIORITY APPLN. INFO.:			US 2002-427980P	P 20021121
			WO 2003-JP14790	W 20031120

AB Disclosed are a peritoneal dialyzate fluid containing ATP or its salt and a peritoneal dialysis method using the same. This peritoneal dialyzate fluid is highly safe and causes no peritoneal injury even employed over a long time. The effect of ATP on protection of human peritoneal mesothelial cells (HPMC) from high concentration of glucose in the culture medium was examined

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT



L26 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1995:992982 CAPLUS

DOCUMENT NUMBER: 124:37763

TITLE: Neutral electrolyte solutions containing sugars and citric acid

INVENTOR(S): Hama, Sumio; Ishihara, Tomoko

PATENT ASSIGNEE(S): Terumo Corp, Japan

SOURCE: Jpn. Kokai Tokkyo Koho, 5 pp.

CODEN: JKXXAF

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	---	-----	-----	-----
JP 07252137	A	19951003	JP 1994-22492	19940221
PRIORITY APPLN. INFO.:			JP 1994-22492	19940221

AB The solns., which contain sugars, pH-retaining agents, and citric acid (I), show pH 6.5-7.5 and are sterilized, are claimed. Sugars are stable in the solns. although pH is neutral and the solns. are useful as washing liqs. for isolated and cultured cell, dialysis solns., and transfusions for parenteral nutritions. An aqueous solution containing NaCl

120,

Na2HPO4 10, NaH2PO4 5, trisodium citrate 5, and glucose 15 mmol/L with pH 7.0 and osmotic pressure 296 mOsm was prepared The solution was useful for washing of blood cells isolated from human blood to show little hemolytic effect and reduction of ATP content.

L31 ANSWER 49 OF 82 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1967:74464 CAPLUS

DOCUMENT NUMBER: 66:74464

TITLE: Dinitrophenol edema. Pathophysiologic model for cerebral edema

AUTHOR(S): Reulen, H. J.; Baethmann, A.

CORPORATE SOURCE: Univ. Munich, Munich, Fed. Rep. Ger.

SOURCE: Klinische Wochenschrift (1967), 45(3), 149-54

CODEN: KLWOAZ; ISSN: 0023-2173

DOCUMENT TYPE: Journal

LANGUAGE: German

AB Ninety-five male albino Sprague-Dawley rats,  $320 \pm 10$  g. body weight, were given 13 ml. 0.45% NaCl by stomach tube 1 hr. prior to the expts. Group 1 was given isotonic glucose solution interarterially, right carotid artery; group 2, glucose + 541mM 2,4-dinitrophenol (DNP); group 3 glucose + DNP and 39.4mM ATP; and group 4 glucose + DNP, but both injected into the tail vein. Two hrs. after the infusion, 0.45% NaCl was again administered and 6 hrs. later the animals were bled to death through the carotid artery. Na<sup>+</sup> and K<sup>+</sup> were determined in the serum. The brain was removed and H<sub>2</sub>O determined by drying at 110°. Na<sup>+</sup> and K<sup>+</sup> were determined photometrically, and frozen brain tissue was deproteinized, homogenized, neutralized with K<sub>2</sub>CO<sub>3</sub>, and the various phosphate compds., lactate, and pyruvate determined according to standard methods. The results showed for group 1 no changes in serum and only a slight decrease in brain K<sup>+</sup>. There was an increase in Na<sup>+</sup> and H<sub>2</sub>O in the brain corresponding to a 4.5% increase in brain volume in group 2. There were no significant changes in H<sub>2</sub>O and electrolytes in group 3 nor in group 4. Changes in brain metabolites after DNP injection into the carotid artery included a 27.5% decrease in creatine phosphate and 21.0% for ATP. ADP, inorg-p- and lactic acid increased. DNP produced edema of the brain, and this edema could be prevented by ATP. 47 references.

ACCESSION NUMBER: 1973:12769 CAPLUS

DOCUMENT NUMBER: 78:12769

TITLE: Oxidation of pyridine nucleotides by membrane structures of the retina

AUTHOR(S): Shukolyukov, S. A.; Zhuchikhina, A. A.

CORPORATE SOURCE: Inst. Evol. Fiziol. Biokhim. im. Sechenova, Leningrad, USSR

SOURCE: Mitokhondrii, Mol. Mekh. Ferment. Reakts., Mater. Vses. Simp. Biokhim. Mitokhondrii, 6th (1972), Meeting Date 1970, 189-93. Editor(s): Severin, S. E. "Nauka": Moscow, USSR.  
CODEN: 25SDAU

DOCUMENT TYPE: Conference

LANGUAGE: Russian

AB In an electrolyte-free medium, the rate of NADH (0.15nM) oxidation by the external segments isolated from the bovine eye retina was higher than that by the mitochondria isolated from the retina, while in a medium containing K phosphate (20 mg/ml), MgSO<sub>4</sub> (5 mg/ml), KCl (30 mg/ml), glucose (30 mg/ml), ATP (2 mg/ml), and hexokinase (0.2 mg/ml), the NADH oxidation by the mitochondria greatly increased and that by the external segments decreased. Na azide strongly inhibited, while cyanide had little effect on the NADH oxidation by the external segments. Amytal inhibited the NADH oxidation by the external segment to a greater extent than that by the mitochondria. Addition of cytochrome c to the Amytal-treated prepns. increased the NADH oxidation

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L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:453073 CAPLUS  
DOCUMENT NUMBER: 140:429098  
TITLE: Peritoneal dialysis method with solution containing ATP  
INVENTOR(S): Kiribayashi, Kei; Yorioka, Noriaki  
PATENT ASSIGNEE(S): Kowa Co., Ltd., Japan  
SOURCE: PCT Int. Appl., 21 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: Japanese  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004045679	A1	20040603	WO 2003-JP14790	20031120
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
CA 2506704	A1	20040603	CA 2003-2506704	20031120
AU 2003284594	A1	20040615	AU 2003-284594	20031120
EP 1563858	A1	20050817	EP 2003-774083	20031120
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK			
CN 1713926	A	20051228	CN 2003-80103652	20031120
NZ 539796	A	20061130	NZ 2003-539796	20031120
US 2006019925	A1	20060126	US 2005-533538	20050502
PRIORITY APPLN. INFO.:			US 2002-427980P	P 20021121
			WO 2003-JP14790	W 20031120

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(FILE 'HOME' ENTERED AT 17:19:29 ON 20 JUN 2007)

FILE 'REGISTRY' ENTERED AT 17:19:44 ON 20 JUN 2007  
E ADENOSINE TRIPHOSPHATE/CN

L1 1 S E3

FILE 'CAPLUS, MEDLINE' ENTERED AT 17:21:32 ON 20 JUN 2007

L2 156348 S L1  
L3 156348 S L1  
L4 438 S L3 AND PERITON?  
L5 62 S L4 AND GLUCOSE?  
L6 2 S L5 AND ELECTROLYTE?  
L7 60 S L5 NOT L6  
L8 0 S L7 AND SODIUM CHLORIDE?  
L9 0 S L7 AND NACL?  
L10 0 S L7 AND SALT?  
L11 24 S L7 AND ACID?  
L12 30 S L3 AND DIALYSATE?  
L13 3 S L12 AND GLUCOSE  
L14 0 S L13 AND ELECTROLYTE?  
L15 0 S L13 AND SALT?  
L16 2 S L13 AND ACID?  
L17 1 S L13 NOT L16  
L18 6 S L3 (P) GLUCOSE (P) ELECTROLYTE?  
L19 1 S L3 (P) GLUCOSE (P) DIALYSIS?  
L20 3 S L3 (P) GLUCOSE (P) DIALY?  
L21 2 S L20 NOT L19  
L22 184 S GLUCOSE (P) DIALYSIS (P) ELECTROLYTE?  
L23 1 S L22 AND ATP  
L24 137 S GLUCOSE (P) DIALYSIS (P) ATP  
L25 8 S L24 AND SALT?  
L26 1 S L24 AND SODIUM CHLORIDE  
L27 47 S L24 AND ACID  
L28 4 S GLUCOSE (P) DIALYSIS SOLUTION (P) ATP  
L29 93 S GLUCOSE (P) ELECTROLYTE? (P) ATP  
L30 11 S L29 AND SOLUTION?  
L31 82 S L29 NOT L30

=> d his

(FILE 'HOME' ENTERED AT 17:53:01 ON 21 JUN 2007)

FILE 'CAPLUS, MEDLINE' ENTERED AT 17:53:14 ON 21 JUN 2007

L1	1 S PERITONEAL INJUR? (P) ATP
L2	0 S PERITONEAL INJUR? (P) ADENOSINE TRIPHOSPHATE?
L3	803 S CELL INJUR? (P) ATP
L4	0 S CELL INJURY? (P) ATP (P) DIALYSIS
L5	0 S PERITONEAL CELL INJURY? (P) ATP (P) DIALYSIS
L6	0 S PERITONEAL CELL INJURY? (P) ATP
L7	0 S PERITONEAL CELL INJUR? (P) ATP
L8	0 S PERITON? CELL INJUR? (P) ATP
L9	0 S PERITON? CELL INJUR? (P) ADENOSINE TRIPHOSPHATE
L10	0 S PERITON? CELL INJUR? (P) NUCLEOTIDE?
L11	169 S CELL INJUR? (P) NUCLEOTIDE?
L12	42 S CELL INJUR? (P) NUCLEOTIDE? (P) TREAT?
L13	0 S L12 AND DIALYSIS
L14	0 S L12 AND DIAL?
L15	63 S CELL INJUR? (P) ADENINE NUCLEOTIDE?
L16	52 S L15 NOT L12
L17	1 S L16 AND PATIENT?
L18	0 S L16 AND ADMINISTER?
L19	106 S L11 NOT L15
L20	106 S L19 NOT L16
L21	75 S L19 NOT L12
L22	19 S CELL INJUR? (P) ATP (P) PATIENT?